In The Specification

Amend page 7 as shown in the enclosed substitute page, in order to clarify the incorporation language:

(Miyako K et al 1997 and 1999). MPP+ destabilizes D-loop structure thereby inhibiting the transition from transcription to replication of mitochondrial DNA (Umeda S. Et al 2000).

Alzheimer's disease patients brains have decreased levels of mitochondrial DNA, increased levels of 8-OHdeoxyguanosine and increased DNA fragmentation (de la Monte S.M. et al 2000). Increased levels of point mutations, for example at nucleotide pair 4366 in the $tRNA^{GLN}$ gene was observed (Shoffner J.M. et al 1993). The risk of Azlheimer's disease increases when a maternal relative is afflicted with the disease (Duara R. Et al 1993, Edland S.D. et al 1996).

DNA damage was proposed as a cause of Lou Gehrig's disease by Bradley W.G. et al and deficiency of cytochrome c oxidase activity and a cytochrome c microdeletion were observed by Borthwick G.M et al (1999) and Comi G.P. et al. (1998).

A decreased activity of mitochondrial complex IV and citrate synthase was observed in Olivopontine Cerebellzr Degeneration (OCD or MSA) (Shapira A.H.V. 1994, 1998).

Biological Actions of Polyamines that Maintain Brain Function and Prevent Neurodegeneration.

However the pathology of several diseases states which are described below involves more than the initial DNA damage and correspondingly the influence of therapeutic agents in these diseases involves control of DNA damage and other cellular injuries simultaneously.

I previously reported the ability of 2,3,2 tetramine in Murphy U.S. Patent No. 5,906,996 to prevent MPTP induced dopamine loss and the applicability of such compounds in the treatment of neurodegeneration, this patent being ~~notated~~ incorporated herein in its entirety by this reference.